It thus appears likely that the effects of nicotinic acid are due to decrease in the production of adrenaline.

SUMMARY

Nicotinic acid in large dosage produces a decrease in eosinophils within two hours, an increase in neutrophils and a slight decrease in lymphocytes. The amide produces eosinopenia at four hours, neutrophilia at four hours and a lymphocytosis at two hours.

Nicotinic acid markedly decreases the ratio of urinary uric acid to creatinine while the amide increases the ratio.

Nicotinic acid acidifies the urine and causes retention of potassium and increased excretion of sodium at four hours. There is a decrease in urinary glycine. The amide lowers urine acidity, increases potassium and sodium excretion and increases the ratio of uric acid to creatinine. There is a marked increase in glycine excretion.

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Case Reports

LYMPH NODE BIOPSY IN THE DIAGNOSIS OF DISSEMINATED LUPUS ERYTHEMATOSUS*

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ENLARGEMENT of the superficial and deep lymph nodes is a prominent finding in the majority of reported cases of disseminated lupus erythematosus. This observation was made in 1872 by Kaposi[1] and in 1899 by Hardaway.[2] Low and Rutherford[3] in 1920 were similarly impressed by lymphadenopathy in their cases.

Clinical suspicion of disseminated lupus erythematosus is most commonly confirmed by finding the lupus erythematosus cell either in the peripheral blood or bone marrow.[4] This case is presented because routine microscopic examination of a lymph node biopsy revealed the histological characteristics which, in the authors' opinion, permitted a diagnosis of lupus erythematosus to be made. A sternal marrow aspiration later revealed lupus erythematosus cells. In view of the fact that, despite clinical suspicion, L.E. cells are not always found, biopsy of lymph nodes may provide positive evidence.

C.R., a Mexican woman aged 31, was admitted to the hospital complaining of joint pains for a period of eight months. In 1946 she had been treated for syphilis with penicillin. There was no further history of past illness. She was never hospitalized except for the birth of two children. Both births were normal full-term deliveries. The family history is negative.

The joint pains that led to her present hospital admission had been migratory in type and had involved all joints at some time. The joint pains had been associated with chills and fever but no sweating. The patient complained of weakness and malaise. There had been no weight loss. For the previous two months she had complained of intermenstrual bleeding. The joint pains had been alleviated by salicylates but never cleared up completely. Other symptoms included myopia, pain in the left ear, tenderness in the epigastrium, and constipation.

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The patient was poorly nourished but well developed and did not appear to be in acute distress. There was a macular rash on the face, not following any particular pattern. The jugular veins appeared distended. Lymph nodes were palpable in the left supraclavicular region. Breathing was regular; lung expansion equal on both sides. The chest was clear to percussion and auscultation. Examination of the heart revealed a soft systolic murmur in the apex. There was a diastolic murmur in the left third intercostal space. The pulse rate was 98; B.P. 120/70 mm. Hg. The liver was palpably enlarged 4 cm. below the costal margin. The spleen also was palpably enlarged 6 cm. below the costal margin.

Diagnosis: Lymph node—changes compatible with lupus erythematosus.

Following this report, preparations were made from the bone marrow and the "rosette" type of L.E. cells was found.

DISCUSSION

Fox and Rosahn[5] have observed œdema and engorgement of vessels as the most prominent histopathological features in the lymph nodes in lupus erythematosus. The lymph sinuses are swollen and distended with lymphocytes, plasma cells and histiocytes. In much of the postmortem material studied, the primary and secondary follicles are absent. The endothelial cells are swollen and hyperplastic. The presence of large neutrophilic to eosinophilic cells, three to four times as large as a lymphocyte and not unlike a megakaryocyte, scattered sporadically through the pulp is considered a constant finding by the above authors. They also refer to the perivascular cuffing by fibrous tissue of the arteries and arterioles—a feature frequently observed around the central arterioles of the spleen. Fox and Rosahn[5] further state that the histopathological changes noted in the lymph nodes are more or less constant and perhaps peculiar to the disease.

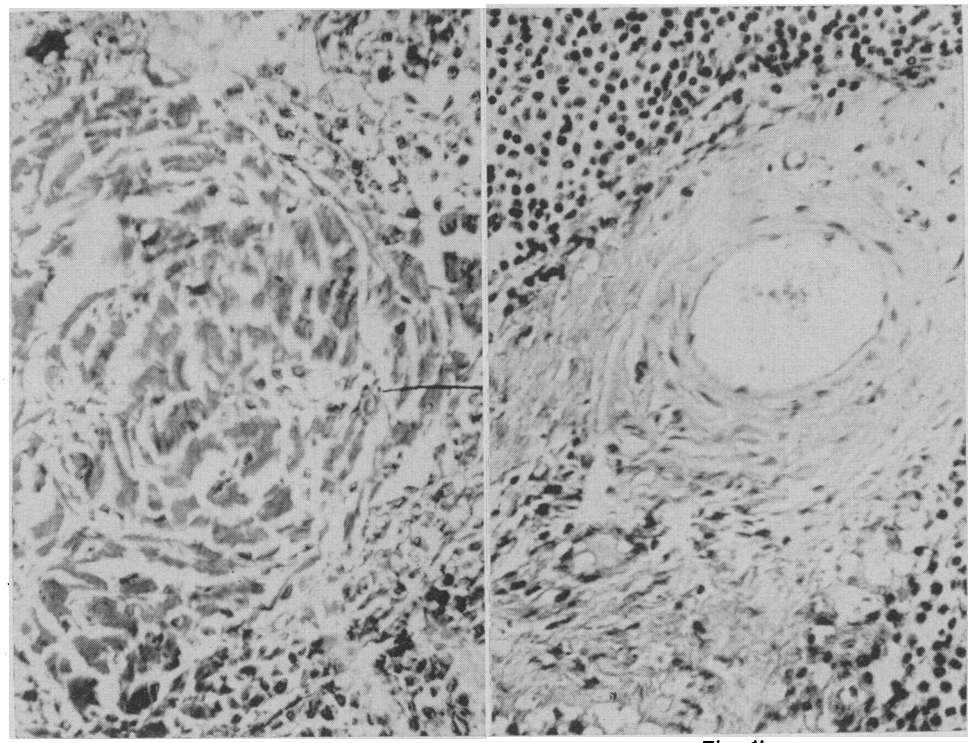

Fig. 1a. Fig. 1b.

Fig. 1a.—Note the marked pericapillary fibrous hyperplasia. The lumen of the vessel is of the usual size. **Fig. 1b.**—Note the marked periarterial fibrosis.

There was fusiform swelling of the joints of the fingers, and the left wrist, elbow and shoulders were tender. The epitrochlear lymph node on the left was the size of a sparrow's egg. Reflexes were normal. Radiological examination of all the joints revealed changes seen in rheumatoid arthritis. Electrocardiographic studies were negative.

The Mazzini test was negative. Blood cultures were repeatedly negative. Urinalysis was essentially negative at all times. The erythrocyte count was 3,800,000; white cell count 5,700; Hb. 10 g.; segmented leukocytes 64; stabs 4; lymphocytes 30; eosinophils 2. The erythrocyte sedimentation rate was 54 mm. Blood non-protein nitrogen 95 mg./100 c.c.; blood urea nitrogen 9.8; uric acid 2.2; A/G ratio 4.4/3.2.

A lymph node 4.5 x 3.2 cm. was examined by biopsy. The capsule was thin and transparent. The cut surface was pale grey, dry and gritty. The consistency was moderately firm. *Microscopically,* vessel changes were most prominent. The small arterioles showed pronounced periarterial fibrosis. The lymphoid tissue was hyperplastic with prominence of the germinal centres. The lymph sinuses were dilated and filled with lymphocytes, plasma cells, histiocytes and reticular cells.

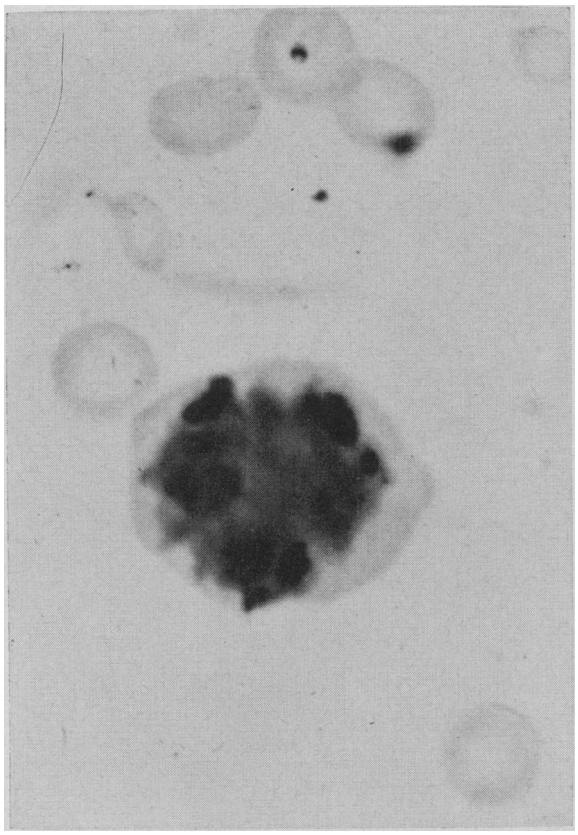

Fig. 2.—Note the rosette type of L. E. cell, the cells being distributed about the periphery of the large monocyte and centrally there is a deep purplish amorphous deposit.

This case report points up the value of lymph node biopsy in cases of suspected lupus erythematosus, if other diagnostic measures have not been fruitful and if an accessible lymph node can be excised and studied.

SUMMARY

This report of a case, in which the diagnosis was based upon the suspicion of lupus erythematosus by virtue of the examination of a lymph node, demonstrates the value of lymph node biopsy in suspected cases of lupus erythematosus since L.E. cells are not always found in such cases. "Histopathological changes noted in the lymph nodes in L.E. are more or less constant and perhaps peculiar to the disease."[5]

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A CASE OF CARDIAC ANEURYSM*

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MANY CASES of cardiac aneurysm are reported in current medical literature. In the majority of these extensive myocardial degeneration had caused general enlargement of the heart, classified as aneurysm. We will confine our definition of cardiac aneurysm to pouches or sacs in the heart wall. The left ventricle is most often involved. Because of the difficulty in diagnosis, cardiac aneurysms are quite frequently recognized only at autopsy.

A case of cardiac aneurysm, although somewhat obscured by lung pathology, is presented. It is of interest that the patient lived fairly comfortably for a period of three and one-half years in spite of the extensive size of the aneurysm. Methods of diagnosis and possible means of prevention are discussed.

Mr. J.C., aged 62, was admitted to the Royal Columbian Hospital on March 1, 1951. He had been well and fit all his life except for brief attacks of bronchial asthma once or twice a year for the previous five or six years. He now complained of severe precordial pain, breathlessness, and being "very wheezy" for a 10-day period. This pain was relieved to a certain extent by heavy drinking. During most of the ten-day period he was up and about, remaining in bed for only brief periods.

On admission, he showed deep cyanosis of the lips, face and fingernails. He was dyspnœic and his respiratory rate was 40. The temperature was 105° F. Clinically, his heart was within normal limits and heart murmurs were not detected. He had a blood pressure of 105/70 mm. Hg. The pulse was 100, feeble and irregular. Auscultation of the lungs revealed mucoid crepitations, particularly at the right base. The white cell count was 12,700; differential count was within normal limits. Erythrocyte sedimentation rate 21. Kahn test negative. Urinalysis essentially negative.

Radiographic examination: Cardiac/thoracic ratio—22.5/30.7. The left ventricle predominates and its outline is very suggestive of aneurysmal dilatation; there is irregular lobulated enlargement of the left ventricle. Bronchial pneumonia is also present at the right base. The electrocardiogram indicated recent anterior myocardial infarction.

The patient made good progress and was discharged from hospital six weeks later. Following his discharge, he had several electrocardiographic examinations. On September 1, 1951, he had an elevation of the ST segment in V4 and V5. as well as QS complex in L1 and all precordial leads from V2-V6. On May 29, 1954, the ST segments returned to isoelectric. On September 1, 1954, there was again a slight elevation of ST in V4 and V5. These examinations were typical of extensive anterior myocardial damage compatible with radiographic diagnosis of ventricular aneurysm.

Second admission to hospital was on May 28, 1954. Since his first admission, the patient had been able to carry on as a plaster contractor, but at times he was

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